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ORIGINAL ARTICLES.

KERATOCONUS, ÆTIOLOGY, AND IMPORTANCE OF EARLY DIAGNOSIS AND TREATMENT.*

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DISCUSSION.

Dr. Alt.—What does the doctor expect from the application of alum, and in what proportion of cases has he seen a decided effect of it on the corneal tissue? I do not understand the possible effect. If it is used strong enough to harden the tissue of the cornea it would interfere with its nutrition and, perhaps, might produce sclerosis with beneficial effect. But I doubt even this possibility.

Dr. Suker.—I would like to ask the doctor if he considers keratoconus identical with keratoglobus, and whether it may not be a congenital condition. I would like to say that it is not always so, nor always binocular. Not much was said about the tension of the eyeball, or whether an iridectomy is of any avail. Would like to ask the doctor about his experience with eserine in these cases.

Dr. O. A. Griffin, Ann Arbor, Mich.—In reference to the correction of refractive errors in this condition, the doctor says that we should not use a mydriatic. It seems to me that in the weakened state of the cornea, the refractive condition should be as carefully estimated as in any other case, espec-

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ially if the patient is young. The spherical aberration which is increased in these cases with dilatation of the pupil, may be counteracted by placing a disc before the eye with a perforation corresponding to the size of the normal pupil, through which the patient sees. An estimate of the refraction made in this manner brings out all the latent defect, with none of the disadvantages of mydriasis.

Dr. Wilder.—When it comes to the point of cauterizing the tip of the keratoconus, it of course means that we will have a scar which will interfere with central vision. I had one experience where I had fairly good results by making a crescent-shaped cicatrix at the base of the keratoconus. The contraction of this cicatrix seemed to cause a flattening of the top of the cone, so that vision was markedly improved.

Dr. Bradfield (closing discussion).—The discussion of this subject and questions asked confirm my supposition and give the opportuny to present to the Academy my views more fully.

My observation is limited to my own private practice, and only having about 8,000 cases to draw from, I frankly admit that my conclusions should not be taken as final, but only ask that they be given their proper place.

In reply to Dr. Alts's question will say, that just how the alum affects the cornea I do not know; but I do know that in the proper cases under its use the ectasia disappears as is demonstrated both by the ophthalmometer and the refraction. One case treated over a year ago in which vision was $^{20}/_{80}$ in the left eye, $^{20}/_{25}$ w.—150◯—1.00 ax. 135°, the ophthalmometer showing the two principal meridians markedly at variance from right angles, now has a vision = $^{20}/_{20}$, accepts a plus 0.75 sphere, and has almost a perfect cornea shown both by the ophthalmometer and the refraction.

In answer to Dr. Suker's question, I will say I do not think keratoconus is ever congenital; I never saw a case before puberty, and do not think it related at all to kerotoglobus.

The conclusions from my observation on keratoconus which I wish to present to the Academy may be summed up as follows:

Keratoconus results from disturbance in the general system affecting the nutrition of the cornea, of which the

nervous phenomena of puberty are the most important. Attention to the constitutional trouble is the most important part of the treatment. While the cornea is in this plastic condition applications of alum will reduce the ecstasia, but after the disease has come to a stasis it will be of no use.

When the ecstasia has advanced till it has become staphylomatous and the center thin, only a radical operation, as cauterization or excision, is of any use.

Many cases of keratoconus recover spontaneously before any perceptible cone results, but owing to the irregular astigmatism left, require special attention in correcting errors of refraction, owing to the different refraction of different portions of the cornea as the pupil varies in size. To be successful, treatment must be begun early and continued till the cornea recovers its proper nutrition and tonicity.

PAMPHLETS RECEIVED.

"A Note on the Histology of Vernal Conjunctivitis" by G. E. de Schweinitz, M.D.

"The Influence of Consanguinity on the Organs of Special Sense," by L. W. Dean, M.D.

"Amber Yellow Glass in the Examination and Treatment of Eyes," by H. H. Seabrook, M.D.

"An Unusual Case of Spontaneous Bilateral Haemorrhage from the Ear," by M. A. Goldstein, M.D.

"An Ophthalmic Clinic in the Hospital of the University of Pennsylvania," by G. E. de Schweinitz, M.D.

"Phlyctenular Ophthalmia in the White and Black Races, with Notes on its Local Treatment," by H. D. Bruns, M.D.

"The Histology of Bulbous Keratitis in Glaucomatous Eyes," by G. E. de Schweinitz, M.D., and E. A. Shumway, M.D.

"Concerning a Possible Etiological Factor in Tobacco Amblyopia Revealed by an Analysis of the Urine of Cases of this Character," by G. E. de Schweinitz, M.D., and D. L. Edsall, M.D.

OPTIC NEURITIS (BILATERAL) COMPLICATING WHOOPING COUGH.*

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A DILIGENT search into the literature has revealed reports of three cases of optic neuritis complicating whooping cough. I have thought it best to give the full report of these cases, in addition to report of my own case, and have also included an abstract of a case of ischemia of the retina, coincident with this disease, for reasons given below.

Alexander's[1] Case II: "Girl, 12 years old (still under treatment), totally blind, Oct. 3, 1887. Whooping cough preceded blindness two weeks and accompanied by intense headaches. On Sept. 15 the girl noticed that everything grew darker, and on Oct. 3 was totally blind. Pupils rigid, no reaction, either consensual or accommodative. Cornea, iris, nothing abnormal, but ophthalmoscope revealed in the fundus of both eyes optic neuritis.

Under treatment blindness decreased, and Nov. 1 was able to count the fingers at a distance of 8 [illegible]s. Middle of Nov., still better and decrease of optic neu[illegible]s. Whether there will be complete recovery, cannot be said on account of advanced stage of atrophy of the optic nerve."

Jacoby's[2] case I: "Girl, 6 years old, consulted with Dr. Hohlich, Nov. 15, 1888. Patient has had different diseases; when two years old, pneumonia accompanied by convulsions. From that time child complained of pain in the occiput. The other children in the family have passed recently through all the stages of whooping cough, and are recovering. For some weeks the child has suffered from spasmodic cough and occipital pains. The day before yesterday the child asked her mother why she remained away so long, as it was getting dark. This aroused the mother's attention that the child was becoming blind. Next day the patient was examined by Dr. Gruening. His findings were, dilatation of pupils ad

*Read before the American Academy of Ophthalmology and Oto-Laryngology at Indianapolis, Ind., April 10, 1903.

maximum; no reaction to light or convergence; bilateral neuritis without hemorrhages in the opticus; sensibility as to light quantitative. Another examination revealed that the vision was limited to right eye and that she was able to distinguish larger objects, like a watch, at a distance of 5 inches. Child examined again on 18th. Reaction of left pupil and larger [illegible]s could be seen with both eyes. Vision improves gr[illegible]. Was normal on Nov. 28. Ophthalmoscopic exan[illegible]tion was negative. Since that time child is all right."

Callan's, P. A.,[3] Case III: "Kate M., aged 11 years; patient undersized and not strong for her years. When 6 years old had a very severe attack of whooping cough, lasting three months. Patient was much reduced by severe whoops and mother feared for her recovery, she was at times so prostrated. At the end of three months of the disease the whoops suddenly ceased, but a very dangerous complication arose, viz., brain trouble. Patient on attempting to walk would become dizzy and stagger, complaining of severe headache and pains in the joints and all over the limbs. Mind wandered at times. Was obliged to remain in bed for three weeks and at the end of that time her headache and dizziness left her, but she could only see very imperfectly. The mother, who was not a very intelligent person, noticed that the child in walking would run against tables and chairs, showing plainly that she did not see well.

"Patient was examined by a very competent oculist, who told the mother that "the eye nerves were swollen" (optic nerves).

"For some months there was improvement in the girl's sight, but this failed her again. At the present time there is well marked white atrophy of both discs. V. R., movement of hands before the face, V. L., fingers at 8 feet.

"Here we have a case in which a long continued attack of whooping cough brought about a passive congestion of the brain, with edema. This led to choked discs and subsequently to atrophy."

Dr. H. Knapp[4] reports a case of retinal ischemia in whooping cough in a boy 3 years old; total blindness, no hemorrhage in fundus or subconjunctival. Dr. Knapp be-

lieved the ischemia due to hemorrhagic effusion into sheathes of the optic nerves, or general anæmia. Paracentesis; improved vision.

Boy died three months later with pneumonia, as had been predicted by Prof. Loomis. This abstract of the case is reported because of the theory of pathogenesis advanced.

Author's Case:* Oct. 21, 1902, Ida B., aged 8 years, came in my service at the Illinois Charitable Eye and Ear Infirmary on account of subconjunctival echymosis of the right eye. Her mother gave me the following history: Four weeks before, the patient contracted whooping cough; has been whooping last two weeks. The coughing seizures, she says, are very severe; has six to eight during the night and fewer during the day. The mother says she is perfectly well excepting when she coughs. Has a good appetite; plays out of door as usual; sleeps well excepting when the seizures occur. She has never had any illness save an attack of measles two years ago, which left no sequelæ. She has never had convulsions. Does not complain of headaches excepting immediately after coughing spells for a short time.

The patient is the youngest of thirteen children, eight of whom are living and well; the others died of "lung fever" and other diseases at different ages. The mother is a well preserved, healthy woman. Father's health is good. The patient is a rosy-cheeked, well developed girl with no discoverable evidence of illness except during attacks of coughing. No motor disturbances to be found; possibly deep reflexes slightly exaggerated. Sense of hearing and smell normal.

Examination of the eye: R. V. $^{20}/_{15}$, L. V. $^{20}/_{15}$. Inspection of the right eye, aside from the subconjunctival ecchymosis, showed dilatation of the pupil, which responded to accommodation and consensually; to direct light very feebly. My colleague, Dr. J. Brown Loring, on making the ophthalmoscopic examination of the fundus, called my attention to the slight blurring of both discs.

Oct. 25. Patient presented herself, having ridden on the street car and walked together a distance of four miles. V. R. and L. $^{20}/_{15}$. Right pupil still dilated and responding feebly to direct light. Optic discs more blurred. Slight

*This case (by invitation) was reported at December meeting of the Chicago Pediatric Society.

amount of exudate in the retina below the disc, obscuring the temporal branch of the inferior branch of the central artery of the retina at one point. Analysis of the urine, both chemic and microscopic, negative. Temperature 99.2; pulse 80. Seems well.

Oct. 30. Ecchymosis gradually disappearing. V. R. and L. $^{20}/_{15}$. Right pupil responds to light better today. Neuritis more pronounced. Patient does not now have nor ever has had double vision. Temperature 99.2; pulse 80.

Nov. 6. No change in condition of the patient, excepting that neuritis is more pronounced, and temperature higher, 100° F.

Dec. 2. V. R. and L. $^{20}/_{15}$. Ecchymosis gone. Pupils respond normally to light and accommodation. Examination of urine, both microscopic and chemic, negative. Neuritis more pronounced. Mother says the child plays and acts in every way perfectly well. Whooping cough subsiding. Temperature same as at last visit, 100 F.

Jan. 15, 1903. Seems well, but has increased temperature, 99.5° F. Pulse 80; coughs occasionally; V .R. and L. $^{20}/_{15}$. Fields for red and green normal. Had difficulty in getting the peripheral fields on account of inability of the patient to appreciate the test; however both seem about normal. Discs still swollen; retinal pigment somewhat disturbed.

Blood count by Dr. E. V. L. Brown, Asst. Pathologist of the Illinois Charitable Eye and Ear Infirmary, and Dr. W. K. Spiece, was made with the following findings: "Reds," 4,966,800; "whites," 10,000. Therefore, whites to reds as 1 to 496.

The blood examination, as well as the general appearance of the patient, show that anemia is not the cause of the neuritis.

March 4. Temperature normal. Pulse 76. V. R. and L. $^{20}/_{15}$. Patient attending school; good appetite; apparently well—however has an occasional coughing seizure. Right disc slightly swollen, but evidently well on in regressive stage. The left disc slightly pale, but no swelling present.

May 20. Swelling of discs entirely gone; a perceptible amount of connective tissue at site of exudate on vessel described above; also a decided deposition of connective tissue in discs V. R. and L. $= {}^{20}/_{20}+$.

Prognosis in this case cannot be definitely known at the present time, but it is altogether probable that good vision will remain.

Analysis of findings in the above cases: A study of these four cases shows that optic neuritis occurs in girls, beginning about the 14th day of the convulsive stage in half the cases, opportunity for observing these cases being good; while in the other two cases the complication followed "after some weeks" and after "four months," the evidence being not so reliable.

Evidence of cerebral trouble—"intense headache" and "severe headache, dizzy, would stagger, mind wandered"—present only in half the cases. Ophthalmoscopic findings of the three authentically reported cases show optic neuritis without hemorrhage in the opticus. In only one case (author's) was exudate in the retina reported. In Dr. Callan's case ophthalmoscopic findings were not given except the expression "swollen eye nerves."

Disturbance in motility of the iris reported in all of the cases; in three vision was greatly reduced, while in the fourth there was no disturbance perceptible.

Perfect restoration of sight followed in one case (Jacoby's), while normal vision is present in author's case and but little disturbance of sight is probable. In Alexander's case, vision improving but no final report made; while in the Callan case white atrophy followed with quantitative vision.

Optic neuritis with and without cerebral complications, as above stated, suggests the probability of the cause not being the same in all cases. In sudden hemiplegia, aphasic disturbances, hemianopsia, etc., coming on during the coughing attacks, modern authors almost unanimously give the credit to "mechanical influences," that is, rexis, with the accompanying hemorrhage into the brain and cerebral meninges, and other circulatory disturbances.

The tetanic expiratory movement which characterizes the coughing attack in this disease, increases the intra-venous pressure to such an extent that rupture of the smaller veins and capillaries occasionally occurs, producing the above results, in the same way that sub-cutaneous and sub-mucous ecchymoses are seen in the skin and mucous membranes. The

optic "nerves" may become involved in such complications when menengitis ensues in the form of a descending neuritis.

In the above four cases reported, three of optic neuritis and one of ischemia of the retina, an attempt at giving the pathogenesis is made by Knapp and Callan only. Knapp explains the case of ischemia of the retina as being probably due to "hemorrhagic effusion into the sheaths of the optic nerves;" while Callan believes that "long continued attacks of whooping cough brought about a passive congestion of the brain with œdema; this led to choked disc and subsequently to atrophy."

In this connection it might be well to mention a case reported by Sebrigondi[5] in which a girl of 6 years is said to have become blind with every coughing spell, produced, he thought, by blood stasis.

A. Steffen[6] reports a girl of 8 years of age, who saw indistinctly during coughing spells and lost some of the sharpness of sight in the intervals while the spasmodic stage lasted.

Infectious influences: The consensus of opinion has not settled upon any particular germ as the cause of this disease.

Pronounced leucocytosis,[7] more precisely speaking, lymphocytosis seems to be the only blood change so far observed.

Pathologic changes in the blood vessels have not been reported, I believe; however, the well known predilection that infectious diseases and toxic states have for the vessels, especially of the nervous system,[8] render it possible that fatty changes occur in this disorder in the capillary endothelium of the vessels of the brain and the brain tract we call the optic "nerve."

This infectious disease is characterized by convulsive or spasmodic manifestations. Whether the infective agent excites the respiratory spasm through central or peripheral irritation or inflammation of the nerves supplying the pharynx, is as yet undetermined.

Peripheral neuritis does occur in this disease. Eschner[9] has collected the reports of seven cases. Three of these, the cases of P. J. Moebius,[10] E. Mackey[11] and M. L. Guinon,[12] unquestionably should be so classified. F. A. Craig[13] reports a case which Eschner believes to be an inflammation of the sixth and seventh nerves.

The optic neuritis in the case I have reported is probably intra-ocular, for the especial reason that there has been no disturbance of central vision, no scotoma for red or green, and for the additional reason that peripheral vision is good. I think it is due to the direct action of the toxins of whooping cough upon the nerve tissue. The on-coming of the inflammation during the most acute period of the disease, the second week of the convulsive stage, and the gradual recession of both the neuritis and the spasmodic cough, might favor either the mechanical or the infection theory. If the cause is mechanical, the disturbance is exerted *through* the veins and not *cutside* of them as in hemorrhagic extravasation or œdema of the optic nerve sheaths, for the reason that there has been absence of symptoms of brain lesion.

Blood stasis is but a temporary symptom, the equilibrium of the circulation soon being established. It would not account for this condition unless chronic local venous engorgement of the nerve head resulted therefrom, accompanied by diapadesis of the leucocytes, etc.

Such a condition can, I think, be better explained as resulting from the influence of the toxins according to Metschnikoff's theory of phagocytosis.

Generalizations derived from the study of four cases can be, at most, only suggestive.

Conclusions suggested:

1—Optic neuritis complicating whooping cough seems to occur most frequently in girls (four cases all girls).

2—Occurs with and without evidences of cerebral complications.

3—Vision may or may not be disturbed.

4—Prognosis as to sight good when no cerebral complications exist.

5—Optic neuritis may result from direct action of toxins of pertussis upon the nerve head.

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DISCUSSION.

Dr. Wilder.—I saw this case when it came into my service at the Illinois Charitable Eye and Ear Infirmary in Chicago. The peculiar feature is that there should be this disturbance about the head of the optic nerve and yet so little functional disturbance of the eye. The central vision was normal and no contraction of the peripheral vision. The patient still has good central vision, so our only conclusion can be that there was not a very severe inflammation, although the appearance of the outline of the disc suggests beginning neuritis. In this case it was probably a slight œdema of the tissues of the disc and not an exudate, because vision was so good and there was no contraction of the visual field. The veins were somewhat engorged and tortuous. It was evidently a case of beginning neuritis; whether due to the toxins of whooping cough or due to the spasms of coughing is a surmise. We do know there is congestion of the vessels of the head in coughing, and we can understand how intraocular hæmorrhages may occur, particularly if the condition of the blood is such as to allow weakening of the walls. Slight œdema of the nerve or retina might be caused in the same way.

Dr. Gamble (closing discussion).—In reply to Dr. Wilder I would say that the exudate entirely obscured the vessel above spoken of, at one point. The vessel is still obscured at this point to a lesser extent than it was three or four months ago. It has the appearance of having become organized into connective tissue. This is also true of the exudate in the heads of the nerves. Suggestions of bands of connective tissue can be seen. A few months later this condition will be easily demonstrated, I think.

NEURASTHENIC ASTHENOPIA.*

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UPON investigation of text-books and literature on ophthalmology, I find the subject of neurasthenic asthenopia is given such sparse mention that one would be led to regard the disease as of very infrequent occurrence, or else one of such comparative insignificance as to be unworthy of much thought or attention. This has all the more perplexed me in view of the fact that my experience has brought me in contact with a considerable number of these cases. The importance of differentiating them from ordinary cases of ametropia or heterophoria at once becomes apparent, especially since we are at first apt to be misled by the patient whose history as told by herself would naturally lead one to the belief that some form of refractive error or muscular imbalance was the primary cause of the symptoms annoying the patient.

The diversity of results obtained, viz., only partial improvement or no improvement at all following the application of glasses or other treatment, led to a further investigation of this class of cases.

The term "neurasthenia" was invented by Beard in 1868. It is generic term applied to all morbid conditions essentially characterized by exhaustion of the nervous system.

According to the predominating phenomena, Regis has divided it into the following forms: cerebral form (cerebrasthenia), the spinal form (myelasthenia), the cardiac form (cerebro-cardiac neuropathy), the gastro-intestinal form (cerebro-gastric and intestinal neurasthenia), and lastly the genital type (sexual neurasthenia). Therefore it is seen that neurasthenia is not a disease but a group of diseases, a sort of diathesis with a most varied symptomatic expression.

The cause, which is essentially hereditary, takes its origin in the different diatheses, viz., neuroses, psychoses, alcoholism, syphilis, tabes, etc, It will be seen therefore, that in the large majority of cases degeneracy forms the ground work for the development of the malady. Also, the disease may be due to accidental causes such as shock, traumatism, etc.

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As occasional disorders we have all the circumstances physiological or pathological, moral or physical, capable of either suddenly or slowly producing nervous exhaustion; puberty, troublesome pregnancies, disorders of the uterus or intestines, typhoid fever, hemorrhage, venereal diseases, sexual excess, mental strain, great fatigue, etc.

From this short description of the disease it can be seen how varied may be the manifestations of the disease. However, there are certain symptoms that are rarely absent, and they have been called by Charcot "neurasthenic stigmata." They are as follows: headache—frontal and occipital, sensation of emptiness of the head, insomnia and disturbed sleep, psychic adynamia, motor enfeeblement, spinal hyperæsthesia, gastro-intestinal atony, genital and vaso-motor disorders.

The symptoms referable to the head are almost invariably present and are usually of an aggravated form. Herein lies the reason for these patients being referred to or seeking the advice of the ophthalmologist.

Should the specialist not pursue his investigation of the case beyond the history of head symptoms as related by the patient, he will naturally be making the same error as was made by the one referring the case.

My experience with these cases has been somewhat as follows: Vision with or without mydriatic is $^{20}/_{20}$, or nearly so. Of course there may be some refractive error, but it is usually way out of proportion to the intensity of the suffering endured by the patient.

It is assumed that a mydriatic is employed as a routine practice in determining the full amount of error of refraction. One of the first expressions of the patient to put us on guard is the assertion that as the examination progresses there is a continued failure of vision, the patient complaining of fatigue and asking for periods of rest. In taking the field of vision we again note another peculiarity, viz., the longer we continue the test the more constricted becomes the field of vision. In endeavoring to accurately locate the axis of astigmatism, we here come upon another characteristic manifestation of the disease. There is a constant shifting of the axis of astigmatism, and this may be true whether actual astigmatism exists or not. The symptoms may point to the presence of

muscular asthenopia, and it is here that another characteristic manifestation of the disease is found. Employing the Maddox rod test, it is found that there is no permanent point of fixation of the streak, and often there is a characteristic swinging movement of the streak similar to the swinging of the pendulum of a clock. This movement may be confined to one side of the candle flame, but frequently it swings rhythmically from one side of the flame to the other. The two last named manifestations, viz., the variable astigmatism and the uncertain heterophoria, with the characteristic to-and-fro motion of the light streak together with the constricted field and early fatigue of the eye under examination, I consider pathognomic of neurasthenia, especially when there is found no refractive error or one so low as to be out of proportion to the intensity of the symptoms.

Color-blindness is said to be present occasionally, though I have never observed it in any of my cases. Patients may complain of dread of light with blepharospasm, lachrymation, neuralgia, etc., symptoms which point to a supersensitiveness of the retina. Oftentimes blurring of vision seems to be the most disturbing feature of the disease, and insomuch as this renders impossible the continued close application to near work, patients complain most bitterly about it.

In a large percentage of these cases, further investigation will reveal the presence of some of the other stigmata as classified by Charcot. In other words the neurasthenic eye, as it may be called, is only a link in the chain of symptoms characterized as stigmata of this disease. These symptoms alone do not constitute neurasthenia, but as in other forms of degeneracy it is the sum total of all the different manifestations which present the typical picture.

Experience has shown that this disease is almost exclusively confined to young females, though I have noticed it a number of times in women undergoing the menopause. Its sister affliction, hysterical amblyopia, is often associated with it, in which case the diagnosis becomes much simplified.

Going beyond the assertion that these symptoms are due to degeneracy, I should regard them as being due to irregular, spasmodic stimulation of the centers controlling the functions involved.

There is no reason why neurasthenics should not be affected with refractive errors similarly to any other class of patients, and often they are greatly benefitted by a prescription for the proper glasses. However, considering the origin of true neurasthenia, which is essentially central, we are not justified in promising or expecting a cure.

Because of reasons given above, the only reliable test in determining the presence or absence of refractive error is found in retinoscopy, and then the full correction should be worn in case some error can be demonstrated.

As a protection to ourselves and in justice to the patient, she should be given a clear understanding of her condition and be dispossessed of the idea that glasses are to be a panacea for all her sufferings. It should be impressed upon her that rest from near work is the most essential element in establishing relief or a cure. Constitutional treatment may be highly beneficial, though vigorous physical exercise in the open air and gymnastics indoors in inclement weather, I think, will be found most efficient in caring for these unfortunates.

DISCUSSION.

Dr. Griffin.—This class of patients is interesting in its symptomology and variability of refraction. I have at present a case under observation which presents many of the symptoms that the doctor has cited. The patient is a professor in the University of Michigan, who through hard work has been rendered very neurasthenic. To point out a few of the peculiar things about him, he imagines that the ingestion of certain articles of food, e. g., rice, produces a torsion of one of the eyes; and at other times a peculiar sensation about the eyes indicates the presence of a fever. Although he has been tested several times for the torsion and rise of temperature, when these conditions have been supposed to exist, I have not been able to substantiate any such conditions. A test both with and without a mydriatic shows an emmetropic eye upon one side and but $^1/_8$ diopter of astigmatism in the other. The muscular condition varies from time to time, and a correction for his presbyopia does not prove satisfactory. He has consulted ophthalmologists of note in the East with

no results. The fact that he is becoming presbyopic may explain some of the difficulty; one day he feels the need of aid in near work, the next not. A great many persons are troubled in that manner when they begin to wear the presbyopic glass. Another peculiar symptom about my case is the fact that he says he sees two lenses before the eyes, and is always conscious of two fields, although the lenses were carefully centered. I hope some one may give me an explanation of this phenomenon. Of course it is not necessary to add that these cases are unsatisfactory to deal with. They must be handled with a great deal of care and patience.

Dr. Bradfield.—I appreciate the paper and wish to ask the doctor if in this class of cases, when the patients insist that they get no benefit from the glasses, he advises they should wear them constantly or at their pleasure?

Dr. Heckel.—This condition is exceedingly perplexing and calls for all the ingenuity a man possesses, inasmuch as the dynamic refraction varies from day to day and from week to week. It taxes the physician's patience and the patient's patience. It may occur at any age. I had a case recently in a gentleman 65 years of age; he consulted every oculist in the city, including myself, without relief. Sometimes, in spite of everything you may do, glasses are of no avail.

Dr. Goux (closing discussion).—Some neurologists say that these cases are practically incurable, and this I think is true in all cases having their etiology in degeneracy. Cases due to other causes are more amenable to treatment and may be cured or relieved by removal of the exciting cause. In regard to Dr. Bradfield's question, I find that even though I have instructed my patients to wear their correction all the time, if I pin them down to an actual statement I find they are not wearing their glasses as instructed. I take the precaution of telling them to put them on when they get up and to keep them on all day. Sometimes it will be quite a long time before a patient will be able to wear a glass and get any comfort out of it. I have had a number of cases where they could not wear the glasses, though they were correct. The variable condition of the patient, dependent upon the condition of the nervous system, makes the conditions so different from day to day that what you might prescribe as proper to-

day might tomorrow not be satisfactory. These patients will visit every oculist in town, and that is why I point out the necessity of making them thoroughly familiar with the character of their trouble. If they are made to understand this is not a local condition but a manifestation of a general disease, they will be much better satisfied.

DEGENERATE OCULAR CHANGES RESULTING FROM CONSANGUINITY.*

By LEE WALLACE DEAN, M.S., M.D.

IOWA CITY.

IN presenting to you a short paper on this very interesting subject my main object is to secure your opinions regarding the various points suggested. I would also like to hear of cases of degenerate conditions about the head in children, the result of consanguinity, encountered in your practice. A search in scientific literature has failed to aid me very much in this line of work.

The question of consanguinity in the first degree in its influence upon the central nervous system has often been discussed; its evil results are so apparent that they have resulted in the prohibition of such marriages in many countries. The general bad results have been so manifest that consanguineous marriages were prohibited even among some of the savage people.[1] The Choctaw Indians are divided into two great septs and no man dare marry in his own sept. The Indians do this because they believe it makes a stronger people. Numerous similar cases could be cited.[2]

Among civilized people consanguineous marriage has been prohibited not only by civil law but by ecclesiastical law.

The question as to whether consanguineous marriage does produce degenerate conditions or not, is very nicely stated by Dr. Talbot.[3]

I am sure there is no question today that if there is a perfect parent stock, and if the offspring are perfect, there can be no bad results from consanguineous marriage. How rare it is, however, to find today such a perfect condition. Liv-

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ing as our ancestors have, in unnatural surroundings, and as we do today, has made a perfect stock a rare thing in the human race.

If on the other hand there is in a family some hereditary taint, the consanguineous marriage of the first degree simply doubles the tendency for the development of the hereditary conditions. Such a condition may have been latent for generations and the marriage of first cousins has so doubled the tendency that several of the children will show the same degenerate conditions. Several of the cases cited later in the paper will illustrate this. Atavism indicates that quiescent factors may be present for many generations that cannot be detected.

With the exception of the central nervous system we find the largest variety of degenerate stigmata in the eye. The reason for this is that the eye is really a specialized portion of the brain and is consequently subjected to the same influences as the central nervous system during its development.

The so-called degenerate stigmata found in other parts of the body are frequently considered to be the result of consanguineous marriage. There is no reason why those of the eye should not be so considered. It might be questioned, however, as to whether some conditions like retinitis pigmentosa ought to be placed in this category.

I became interested in this subject because, when several years ago I was investigating the degenerate conditions of the eye, I found a history of a consanguineous marriage of the first degree of the parents of many of the degenerate children.

Because a child has one or two degenerate conditions it is not considered a degenerate. These conditions are so common that the individual is only considered as a degenerate when several stigmata can be found. The number required differs in different schools.

Some of my cases have been so interesting to me that I take the liberty of mentioning them.

Case 1. Male, age 17. Parents, grandparents, uncles and aunts had no serious eye trouble or degenerate stigmata. Parents are bright, well-to-do people. Has three sisters, Bessie, age 15, who is partially blind; Effie, age 9, whose vision is good; and Grace, age 4, who is blind. Here are

four brothers and sisters, three of whom are partially or totally blind. He has three cousins, one boy and two girls, all in the same family. Of these, Louis, age 16, is almost blind; Oran, age 10, cannot count fingers; and the girl, age 13, seems to have unimpaired vision. The trouble in each case was retinitis pigmentosa. The fathers of the two families were brothers and the mothers sisters, and the fathers were cousins of the mothers. As a result of these two marriages we have in one family four children, three of whom are practically blind, and in the other three, two of whom are partially blind.

Case 2. Female, age 14. Father and mother have brown hair. Parents were related. There is no history of malformation or other degenerate stigmata in the family. The girl is very large but weak; she is not intelligent. Her hair is white; iris is a light blue. Examination of the eyes revealed nystagmus V=$^{6}/_{24}$. Fundi without pigment. Diagnosis, albinoism.

Case 3. This is not a single case but the report of a family with six children. The father, Mr. V., and the mother were both dark complexioned. There was no history of any degenerate condition in the family. They were first cousins. They had but six children. Of these, two were dark complexioned and had good vision; the other four were albinos and had a vision V=$^{6}/_{12}$ or less.

Case 4. Female, age 16. Parents second cousins. Patient's teeth were imperfect; she had but three upper incisors, one root germ not having developed. An examination of the eyes revealed V= fingers in 2 m. Her eyes were so small that she had to hold her lids open with her fingers in order to see. On the right side there was a congenital absence of the iris. In the left eye the cornea is elliptical, being 3 mm. wide in a horizontal direction and 2 mm. in a vertical direction. On this side there is a large coloboma of the iris, one-half of the lower portion being absent.

Case 5. Male, age 17. Parents are perfectly healthy. None of his ancestors have shown any signs of degeneracy so far as could be learned. His father and mother were first cousins. He has one brother and two sisters. One of his sisters is an idiot. She has microphthalmus, misplaced ears, and other signs of facial degeneracy. The patient is exceed-

ingly simple minded. His ears are large and placed at right angles to the head The helix is deformed. His face is covered with a growth of silky hair. The lower jaw is exceedingly small and retrusive. He has no adenoids but always keeps his mouth open. Examination of the eyes reveals on the right side a microphthalmic eye with a coloboma of the iris and choroid. On the left side there was an apparent absence of the eye. There were small lids and a socket. No eye could be felt by introducing the finger in between the lids and feeling about in the orbit. The lower part of the socket was deformed. The union between the malar and superior maxillary bone had never taken place. There was a space one-fourth of an inch wide between the two bones. The patient was given an anæsthetic and a dissection of the contents of the orbit was made. The fissure between the malar and the superior maxillary bone was found to extend back to the spheno-maxillary fissure. In the apex of the orbit lying closely against the optic foramen was found a rudimentary eye. It was removed. It was about one-third of an inch in diameter. With the naked eye no change indicating the cornea could be detected. The eye was hardened in formaldehyde and alcohol and stained with hæmatoxylin and eosine. The eye was composed of an outer thick fibrous coat. The cornea could only be differentiated microscopically from the sclera by the absence of an inner lining of pigmented cells. The eye was filled with myxomatous tissue much more solid than the normal vitreous. It contained some blood vessels, one of which was very large. No trace of retinal elements could be found. The internal structures seemed to have all undergone a myxomatous and mucoid degeneration. The lens and retina seemed to be absent.

Case 6. Female, age 5. No history of any serious eye trouble or of degenerate conditions in family. Parents were cousins. Child was very dull. Left eye began to enlarge shortly after birth. O. D. apparently normal, O. S. very much enlarged. Tension normal. Diagnosis: congenital glaucoma, Left. The left eye was enucleated. The usual cupping of the disc was present. Microscopical examination failed to reveal any obstruction of the canal of Schlemm.

Case 7. Female, age 6 months. I saw in consultation

with Dr. Cooling of Wilton Junction, Iowa. Parents were healthy; no history of any tumor or eye trouble in the family could be elicited. Parents were first cousins. The parents said that two months before the right eye began to increase in size, and a few weeks later the left began. The child apparently could not see. Pupils were dilated; would not react to light. Tension in right eye +2, and in left +1. Both eyes were enlarged, the right the more. Lying behind the iris in each eye could be seen a large tumor. Diagnosis: double sided neuro-endothelioma. Four months after the child was seen it died.

Case 8. Male, age 4. Parents' and grandparents' history negative. Parents cousins. Boy has congenital coloboma of iris in each eye and anterior polar cataract in the right eye.

Of 181 children in the Iowa College for the Blind in 1900, nine, or about 5 per cent., were the result of consanguineous marriage of the first degree. The number of consanguineous marriages of the first degree in the state of Iowa is far below ½ per cent. I ought to say that it is exceedingly difficult to secure a history of consanguineous marriage when present. There were probably others present the result of consanguineous union.

If we exclude from the list those blind children who were blind because of blennorrhœa neonatorum, sympathetic ophthalmia, trachoma, etc., and consider only those who suffered because of congenital conditions, we would find that 14 per cent. were the result of consanguineous marriage of the first degree. These figures seem large. I do not think they ought to be considered as indicating the relative proportion of degenerate eyes in families the result of consanguineous marriage and in those not, because we are considering here only one field. Among the pupils who have entered the college since 1900 the per cent. is about the same.

The tendency for the increase of eye trouble amongst civilized races because of excessive use of the eye has had a tendency to increase the bad results of consanguinity on the eye. The Indians have very little eye trouble. About 40 per cent. of the civilized people have eye trouble due to disease from use. This may be a beginning change in the eye for the better. Fuchs believes that the individuals with the school

myopia have their eyes adapted to their work and that the eye is superior to an emmetropic eye. The eye is not perfect, it is changing. The socket is becoming less deep and the superciliary ridges less marked.

The influence of consanguinity on retinitis pigmentosa has been mentioned by several. Dr. Liebreich says that of 26 cases of retinitis pigmentosa, 53.8 per cent. were the result of consanguineous marriage. Magnus reports 33 per cent. of cases of retinitis pigmentosa in children the result of consanguinity. Of 66 cases reported by Chipault, 45 per cent. were the result of consanguineous marriage. Of 18 cases under my observation where the history of the parents could be secured, eight, or 44 per cent., were the result of consanguinity.

Retinitis is a disease that has a great inheritability. I have under observation a grandfather, mother and son, each having the disease. As it is a comparatively rare condition, there is not the chance for doubling the tendency of its appearing by marriage of individuals not related as the more common conditions.

A very careful examination into the history of the ancestors of the cases I mentioned failed to reveal any eye trouble of any importance. Yet in the first group mentioned we have these cases appearing by intermarriage in the two groups when consanguinity took place.

Albinoism is considered by zoologists to be a degenerate condition. Davis[7] says that consanguineous marriage leads to albinoism. Certainly a large per cent. of albinos are the products of consanguineous marriages.

As to whether consanguinity may play an important part in neuro-endothelioma and congenital glaucoma can only be determined by hearing from more cases. The cases of microphthalmus coloboma of iris, congenital cataract, and anophthalmus mentioned are examples of arrest of development. It is interesting to note that the conditions of the eyes are just the opposite to those of the eyeless fishes and worms that have lost the use of eyes that were in their ancestors perfectly developed, owing to generation after generation living in the dark. Wagenmann[8] has found that in these the lens and retina are almost the least of the structures acted upon. That the phyletic degeneration does not follow the reverse

order of development. None of the adult degenerate eyes resemble stages of past adult conditions. In the degenerate eyes, however, we frequently find the eye in one of its developmental stages.

The condition of the eye is not due to local conditions but is due to some central disturbance. With our present knowledge of the physiology of the central nervous system, one cannot say as to whether there are developmental centers—that is, centers which control the growth of parts—or whether there are simply the trophic centers which control their nourishment. The condition is due to a disturbance of the trophic center, or of both if they exist. One may expect these degenerate conditions in the products of consanguineous marriage, because of the increase of some hereditary tendencies in the germ.

By[9] means of the nuclear division and formation of the second polar body, the excessive accumulation of different kinds of hereditary tendencies or germ plasms is prevented. With the removal of the second polar body, as many different kinds of idioplasm are removed from the egg as will afterwards be introduced by the sperm nucleus. If the sperm nucleus contains the same hereditary tendencies as the ovum there will be a greater tendency for these tendencies to become manifest than if the latter were different.

[1] Algonquins, Iroquois, Delaware, Canadian Indians. Huth's Marriage of Near Kin, p. 92.

[2] Huth, p. 93.

[3] Talbot, Degeneracy; Its Causes, Signs, and Results, p. 79.

[4] Liebreich, Deutsche Klinik, Feb. 9, 1861.

[5] Chipault, Etudes sur les. Mar., pp. 58, 59.

[6] Magnus, Die Blindheit.

[7] American Medical Bi-weekly, Vol 12, No. 13.

[8] Archiv für Entwickelung Mechanik der Organe der Menschen, Vol. 8, No. 4.

[9] Ribot, Heredity, p. 366.

DISCUSSION.

Dr. Vail.—This is a very interesting subject and one that is very important. I rise to report that out of seven children in two families where the parents are cousins, I have found five who had congenital cataract. Of the remaining two, one had slight hypermetropia and the other had

hypermetropia of six or seven diopters. In one family there were but two children. The first, a baby, had cataract and the eyes were operated on in the usual way. I enquired of the mother whether she and her husband were blood relatives. She denied that they were. A year and a half later another baby with congenital cataract was brought by the same mother. I asked again if she and her husband were not related, and she confesssed it was true, they were first cousins. She said her husband told her the other time she must deny it, for fear of trouble in the courts. I felt it a duty at that time to tell her that I considered it a social crime for her to bear any more children by this man, and so far as I know they have had no other children.

Dr. Alt.—I had occasion to observe congenital cataract in two children out of four in a family where the parents were first cousins. At the same time I also operated on the grandmother for senile cataract. This was, I think, an additional proof that it was due to consanguineous marriage. I have operated on a number of occasions in a large family in southern Illinois who have, for I do not knowhow long, intermarried. There is a large number of cases of senile cataract and also congenital cataract in the family. The members of this family are so much accustomed to this condition that one of them, who is a physician, comes to me every few months to have me look at his lenses and see if they are beginning to show cataract formation. He told me that his uncle whom I operated on last was, if I am correct, the 43rd case in the family that had been operated upon for cataract.

SARCOMA OF THE CHOROID.*

By W. STANLEY SAMPSON, M. D.

LANCASTER, OHIO.

THE clinical history of intra-ocular tumors published by von Graefe in 1868 was so complete and exhaustive that little remained to be accomplished by his followers. It is not the aim of the author to break the seal placed upon the subject by the illustrious savant, but to content himself with an epitome of his classical monograph. Sarcoma of the choroid is considered a rare disease, and occurs most frequently between the fortieth and sixtieth years, seldom occurring in childhood—differing in this respect from glioma.

Four stages of the disease are distinguished. From the small tumor of the first stage, recognized only by ophthalmoscopic examination by detachment of the retina, to the second stage—that of increased tension—the transition may be sudden. The symptoms of this stage correspond so completely to those of inflammatory glaucoma, that a correct diagnosis is made with the greatest difficulty, and in many cases not at all. Pain is a cardinal symptom in this stage of the disease, and usually sounds the first alarm of trouble to the patient.

The third stage consists in the tumor passing though the sclera and its growth upon the outside. At this period the pain ceases, and the orbital cavity is filled more or less rapidly, depending upon the point of exit. If the sclera is ruptured posteriorly, the ball is pushed forward, and the growth is longer in making its external appearance than were the rupture anterior or lateral. The growth is now more rapid and the tumor may become as large as the fist.

The fourth or metastatic stage of malignant tumor of the choroid is primary in nearly every case, but generalization of the tumor by the development of metastatic nodules in other parts of the body may occur, and especially in the liver.

"Manz reports a case involving both eyes, the original growth developing in the breast."

The prognosis is grave and always proves fatal if the eye

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is not removed early. Berry gives a patient with sarcoma of the choroid, unmolested, five years to live. Of the 285 cases reported on by Fuchs, 13 per cent. recurred, and most of them in a year.

The treatment is enucleation as soon as the diagnosis is certain that the growth is confined to the ball, and exenteration if the orbital cavity is invaded and the whole growth can be removed.

Mrs. M. A. H., 67 years old, farmer's wife, family history

FIG. 1—Sarcoma of the choroid. Growth actual size. The right hand portion of the tumor contains the eyeball; lachrymal gland resting on top.

good, consulted me on Sept. 20, 1901. Seven years ago she suffered severe pain in the right eye, and was treated by her family physician for neuralgia, but without benefit. One year later she became blind in that eye. The pain suddenly ceased, and some time during the winter of 1900 she noticed a growth appearing at the inner canthus of the right eye. On examination I found a dark red mass, filling the orbital cavity and projecting far beyond the frontal eminence. The eyeball formed the outer portion of the growth, and by careful inspection indistinct traces could be seen of the iris and pupil. The growth was immobile and quite firm to the touch. On Oct. 2, 1901, I exenterated the contents of the

orbital cavity. The growth after removal measured 5 x 5 x 7 cm., was highly pigmented and vascular. (See figure). The optic nerve and the muscles were consumed in the sarcomatous process, excepting the external rectus, which was held by a few remaining fibres to the optic foramen. The remaining portion of the optic nerve was caught by forceps and severed by curved scissors as far back as possible. After a thorough curetment the cavity was dusted with nosophen and packed with sterile gauze. The repair process was rapid and at the end of ten days the patient was discharged. There was no recurrence of the disease, but the patient died fourteen months later from metastatic involvement.

According to the patient's statement, pain preceded blindness in the eye one year, differing in this respect from the typical clinical history found in the text books on ophthalmology. A section through the growth bi-secting the eyeball, gives a clear idea of the point of rupture. A microscopical examination of a section made from the lachrymal gland shows round and spindle cells, pigmented. A section from the optic nerve shows a predominance of spindle cells.

Only recently my attention was called to the following interesting case: C. T., aged 26; excellent family history. When 15 years of age he was struck in the right eye with an arrow, made from an umbrella wire, thrown from a crossbow in the hands a playmate. There was no perceptible wound inflicted, and the eye caused no trouble until two years after, when he noticed that vision began to fail in that eye. Five years ago dark nodules began to appear on the superior surface of the ball, which seem to have pushed their way directly through the sclera. The growth is firmly attached to, or in other words is a part of, a tumor mass filling the eyeball, and when the nodules are viewed separately resemble coffee grains. The relationship of the iris is not interfered with, but the growth can be seen in close proximity to the iris by means of a strong lens and oblique illumination. The patient has been totally blind in this eye for two years. At no time has he experienced pain in or about the eye, and there is not the slightest tenderness on pressure at this time. This case has every symptom of sarcoma, save pain, which, as above stated, never was present. Glioma can

be excluded by the patient's age and the pigmentation. We do not purpose going into the etiology of this disease, but consider the pathologic lesion in this case produced by trauma. Operation was refused and our diagnosis cannot be verified by microscopic examination, but the clinical history is at such a variance with that of the usual case that we venture this report.

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DISCUSSION.

Dr. Vail.—Within the past six months I have removed the entire contents of the orbit, including the eyebrow, eyelids, lacrimal gland, sac—in fact all the orbital structures, and while it seemed a most heroic operation, it was one of the easiest I ever attempted. I was surprised to find out how easy it was to shell out the orbital contents. The incision was simply carried around the outside to include the whole neoplasm, down to the bone, through the periosteum; then by means of the raspatory the periosteum was stripped up and followed around. It stripped off so easily it almost fell out. You just sweep around with your periosteotome, like taking a pie out of a pan, then with the scissors clip off the impacted mass of tissues in the apex of the orbit.

Dr. Alt.—If I understood rightly, the author said that in the secondary stage there is always detachment of the retina. Twenty-five years ago this was held to be correct, but it has been proven untrue. In some cases there is detachment of the retina, in others the retina and growth are grown together, and consequently no detachment can occur.

Dr. Bradfield.—I wish to suggest that in cleaning out the orbit it is always safest to have the thermal cautery handy when the apex is reached. I would ask the doctor how he covers the orbit after the operation.

Dr. Vail.—In regard to the dressing, I simply packed the orbital cavity. The hæmorrhage was insignificant. It commenced, but we put in a packing of bichloride gauze, and

the hæmorrhage stopped. I would hesitate to use the galvanic cautery or the chromic acid in the apex of the orbit, as I regard either to be dangerous—it is so close to the brain. It was the purpose of the after-treatment to be especially careful not to introduce germs from the outside. The dressing was of bichloride gauze, left for several days. Granulations gradually formed and it took four or five weeks for the periosteum to form and epitheliate. It was not necessary to do plastic surgery.

Dr. Wilder.—It is easy to strip off the periosteum, as Dr. Vail says. I feel better satisfied, as Dr. Bradfield says he does, when I have the cautery with me. In one case I had severe hæmorrhage. By gently touching the bleeding point with the galvanic cautery at a dull red heat you can sear these vessels perfectly, and I do not think such treatment is dangerous. Sometimes these cases, where exenteration of the orbit has been performed, will take a long time to heal. I have taken long ribbon strips of Thiersch grafts and with them lined the orbit, thus concluding the case more promptly. By waiting for the new epithelium to form, the healing process is much longer, and it seems to me there is greater danger of recurrence in the low grade granulation tissue.

Dr. Sampson (closing discussion).—It is hard to describe this operation, but it is very easy to do, as Dr. Vail says. It was necessary to make a canthotomy on account of the size of the growth. In this case we controlled the hæmorrhage with sterile gauze. Skin grafting was not necessary in this case. I was prepared for hæmorrhage, but there was no more than we could expect in enucleation. If the lids are in a healthy condition, it is advisable to save as much as possible.

OBITUARY.

SIMON POLLAK.

On Saturday, October 31st, Dr. Simon Pollak, the Nestor of the St. Louis ophthalmologists and aurists, died at his home in St. Louis, at the ripe old age of almost 90 years. Born in Bohemia, he studied medicine at the University of Vienna, Austria, from which he graduated in 1835. A few years later he came to America to practice his profession and had soon acquired a lucrative practice so that in spite of severe pecuniary losses, due mainly to the change in the course of the Mississippi River, he was able to return to Europe for further studies. During these years he chiefly paid attention to diseases of the eye, especially at Vienna, Berlin and Paris. Thus he was extremely well equipped at the period at which he settled down in St. Louis, in 1845. In consequence, he soon enjoyed a large and lucrative general practice with ophthalmology and otology as specialties of preference. He is said to have used the plaster of Paris bandage as the first in America. He is given credit by the best alienists of this country for having introduced the humane treatment of the insane now in use. He was one, and probably the chief one, of the founders of the St. Louis School of the Blind, and in 1860 started the first Eye and Ear Clinic in St. Louis, in what is now the St. Louis Mullanphy Hospital. This Clinic he attended with regularity and unswerving interest and zeal until the day of his death. While not a frequent contributor to medical literature, he has published a number of articles on various subjects, some of which may be found in Knapp's Archives and this Journal. In his younger days his influence in the local medical world was undoubtedly a great and beneficial one. The honorable positions held by him during his long career have been numerous. He had a keen thirst for knowledge and a clear intellect up to the last. His charity was untiring and his death is a sore loss to many outside of his family.

ALT.

BOOK REVIEWS.

LESSONS ON THE EYE, for the use of the undergraduate students. By F. L. HENDERSON, M.D. Third edition. Philadelphia, 1903: P. Blackiston's Sons & Co.

This modest little volume, evolved from two previous private editions, has served the author as a guide for his students. It is concisely written and profusely illustrated, and may well be recommended to students.

TEXT-BOOK OF DISEASES OF THE EYE, for students and practitioners of medicine. By H. F. HANSELL, A.M., M.D., and W. M. SWEET, M.D., with chapters by C. R. HOLMES, M.D., C. A. WOOD, M.D., D.C.L., and W. REBER, M.D. Philadelphia, 1903. P. Blackiston's Son & Co. Price $4.00.

This is another beautiful and excellent text-book on diseases of the eye, the number of which seems to be growing almost too fast. That the subject matter is treated in a masterly way goes without saying, as it comes from the pen of such well-known authors and teachers. It is printed in excellent large type and its illustrations are excellent throughout. It is bound to have a wide circulation.

THE BLOODVESSELS IN THE LABYRINTH OF THE EAR. By G. E. SHAMBAUGH. Decennial Publications of the University of Chicago.

The author has carefully studied the blood supply of the labyrinth of sus scrofa domesticus. He was most careful and painstaking in his investigations which are laid down in this pamphlet, supplemented by beautifully executed illustrations. Every one interested in the anatomy of the ear should study this most interesting little volume.

ALT.

PAMPHLETS RECEIVED.

"Nephritic Eye Lesions," by W. O. Nance, M.D.

"A Pharyngeal Aneurism," by C. W. Richardson, M.D.

"Keratosis of the Pharynx," by C. W. Richardson, M.D.

"Report of a Few Mastoid Cases," by L. R. Culbertson, M.D.

"Purulent Ophthalmia of the New-Born," by W. O. Nance, M.D.

"Operative Treatment of Abnormal Tonsils," by J. A. Donovan, M.D.

"Ocular Manifestations in Chronic Bright's Disease," by G. E. de Schweinitz, M.D.

"Congenital Cyst of the Eyeball; Microscopical Examination," by Arnold Knapp, M.D.

"The Treatment of Chronic Suppuration of the Middle Ear," by J. F. McKernon, M.D.

"The Extirpation of the Lachrymal Sac; its Indications and Technique," by Arnold Knapp, M.D.

"The Importance of the Surgical Treatment of Chronic Middle-Ear Suppuration," by E. B. Dench, M.D.

"Occlusion of the Superior Temporal Artery of the Retina in a Young Anemic Girl," by G. E. de Schweinitz, M.D.

"A Case of Localized Tuberculosis at the Head of the Optic Nerve; Microscopic Examination," by Arnold Knapp, M.D.

"Gumma of the Iris and Ciliary Body with Histological Study of the Enucleated Eyeball," by G. E. de Schweinitz, M.D.

"Concerning the Terms: Antimetropia and Anisometropia Brachymetropia and Hypometropia in Place of Myopia; Hypermetropia and Hyperopia," etc., by G. F. Suker, M.D.